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Other full case

Acute myocardial infarction caused by paradoxical embolism with concomitant pulmonary embolism

Aye Hline, Nikesh Malik, Azhar Khokhar, Rajesh Aggarwal

Cardiology, Essex Cardiothoracic Centre, Nethermayne, Basildon, Essex, UK

Correspondence to Dr Nikesh Malik, nikeshmalik@hotmail.com

Summary

An 86-year-old lady presented with an acute inferior ST elevation myocardial infarction. The coronary angiogram showed a thrombotic occlusion of the right coronary artery, which was aspirated, but there was no underlying lesion to stent. The patient remained very hypoxic on the ward with clear lung fields and was found to also have multiple pulmonary emboli on CT pulmonary angiogram. Paradoxical embolism was suspected as the cause of myocardial infarction and subsequent echocardiography studies indeed confirmed the presence of a patent foramen ovale. Although rare, this mechanism should be considered in patients presenting with acute coronary syndromes without significant underlying coronary artery disease.

BACKGROUND

This case report illustrates the phenomenon of myocardial infarction caused by paradoxical embolism through a patent foramen ovale (PFO). Although rare, it should be considered in patients with acute coronary syndrome with no significant underlying coronary artery disease.

CASE PRESENTATION

An 86-year-old lady presented with central chest tightness for 2 h associated with nausea and sweating. She was previously fit and well with no history of ischaemic heart disease. Her only medical history included a well-controlled hypertension. She had been experiencing breathlessness for the preceding 2 weeks, for which she was undergoing outpatient investigations. Clinically, she appeared very unwell with bradycardia and hypotension.

INVESTIGATIONS

The 12-lead ECG showed intermittent complete heart block with ST elevation in the inferior leads. She was immediately transferred to the cardiac catheterisation laboratory for primary coronary angioplasty.

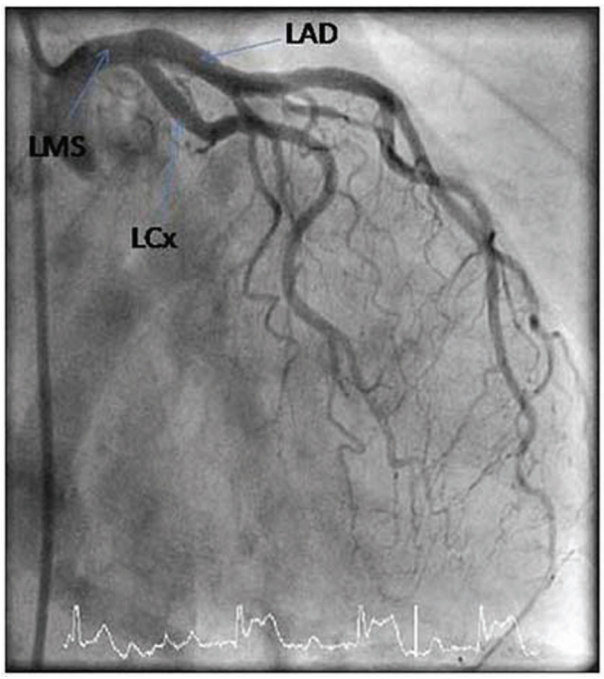

Figure 1 Left coronary system demonstrating no significant disease.

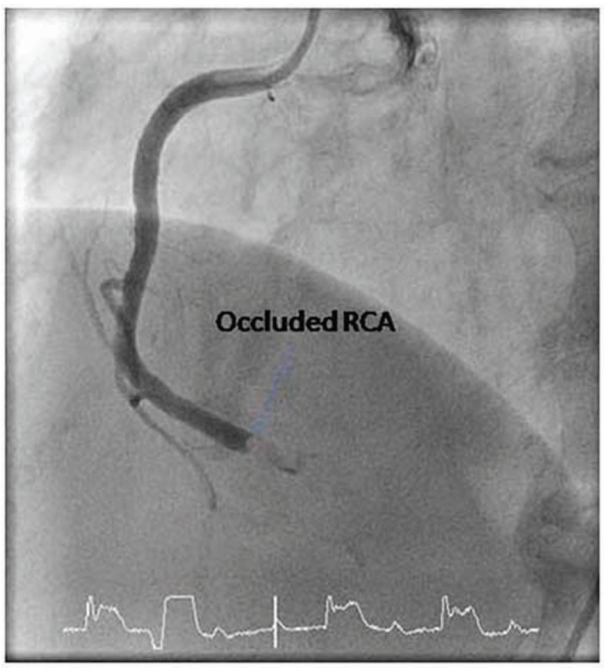

Figure 2 Right coronary artery blocked in the middistal region. ECG showing complete heart block and ST elevation.

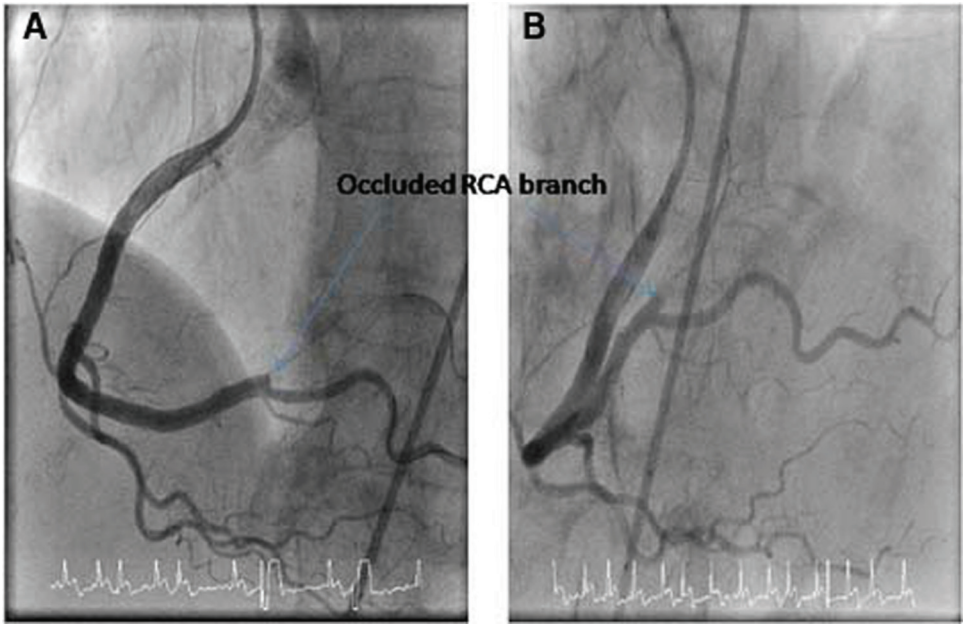

Figure 3 (A and B) Right coronary artery after thrombus aspiration and abciximab bolus. ECG shows reversion of complete heart block and resolution of ST elevation.

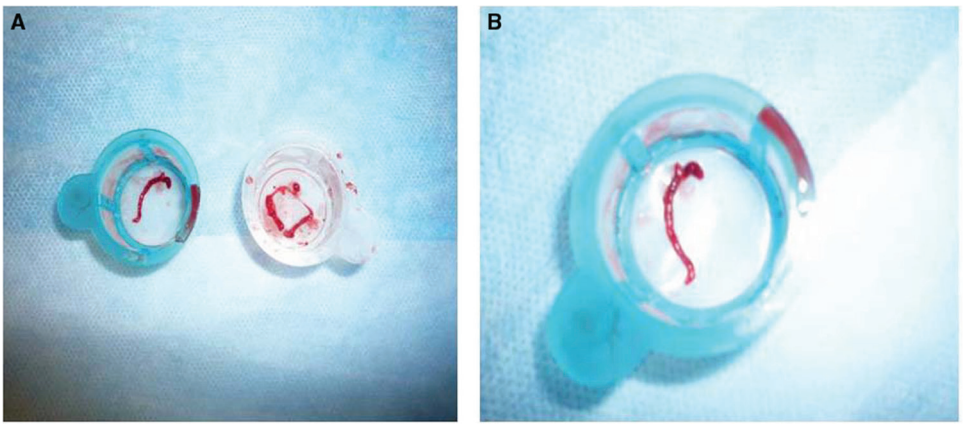

Figure 4 (A and B) Clot aspirated from right coronary artery.

Coronary angiography was performed via the right femoral artery with a 6 French sheath. A right femoral venous sheath was also inserted for temporary pacing if necessary.

The coronary angiogram, shown in figures 1 and 2, revealed normal left main stem (LMS), left anterior descending (LAD) and left circumflex (LCx) arteries. There was an acute thrombotic occlusion of the right coronary artery (RCA) in the mid to distal region.

TREATMENT

A guide catheter was used to cannulate the RCA; intracoronary heparin and abciximab (a glycoprotein IIbIIIa inhibitor) were infused. A guide wire was advanced to the distal vessel and a thrombectomy device was used several times to aspirate thrombus. Full flow was restored, but as there was a significant amount of remnant clot, another device was used to perform further aspiration. There was embolisation of thrombus to one of the distal branches of the RCA, which was also wired and further aspiration was carried out. Despite several attempts, the flow in this branch remained poor (figure 3A,B). Surprisingly, the underlying artery was found to be clear of disease and therefore no coronary stents were deployed.

OUTCOME AND FOLLOW-UP

The ST segments normalised and the rhythm returned to sinus. The patient was pain free and haemodynamically stable at the end of the procedure.

The clot aspirated is shown in figure 4A,B.

Despite resolution of chest pain, the patient remained breathless and was persistently hypoxic in the ward. Arterial blood gas showed type 1 respiratory failure with a Po_2 of 7.4 mm Hg. There was no clinical or radiographic evidence of pulmonary oedema and a transthoracic echocardiogram (TTE) showed preserved left ventricular

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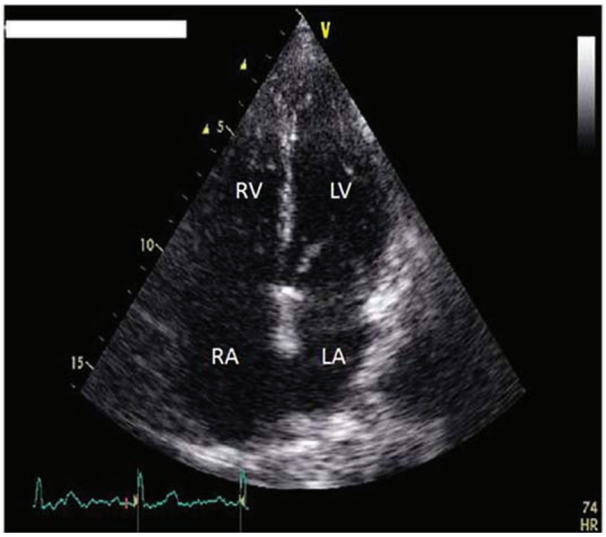

Figure 5 Transthoracic echocardiogram four-chamber view shows dilated right heart. RA, right atrium; RV, right ventricle; LA, left atrium; LV, left ventricle.

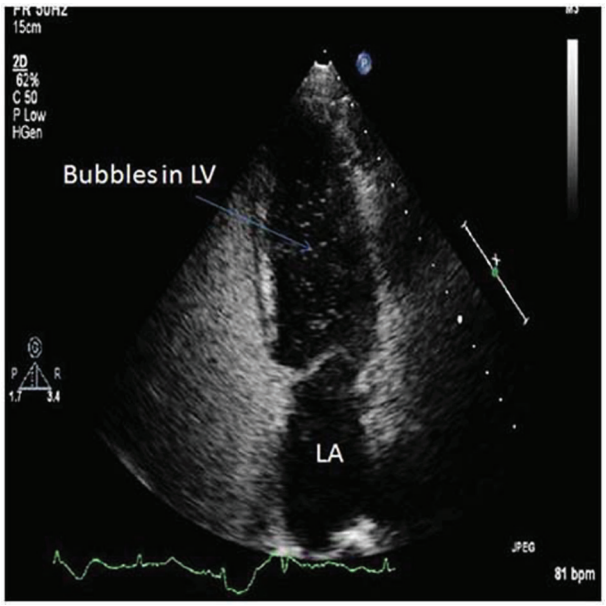

Figure 6 Bubble study two-chamber view shows bubbles in left heart after valsalva manoeuvre.

systolic function with hypokinaesia of the inferior and posterior walls. Interestingly, the right heart was markedly dilated with impaired right ventricular systolic function (figure 5). There was severe pulmonary hypertension, with an estimated right ventricular systolic pressure of 90 mm Hg and a subsequent CT pulmonary angiogram confirmed our suspicion of coexistent multiple bilateral pulmonary emboli. Anticoagulation was commenced.

Given that our patient had presented with an ST elevation myocardial infarction with normal underlying coronary arteries and concomitant pulmonary emboli,

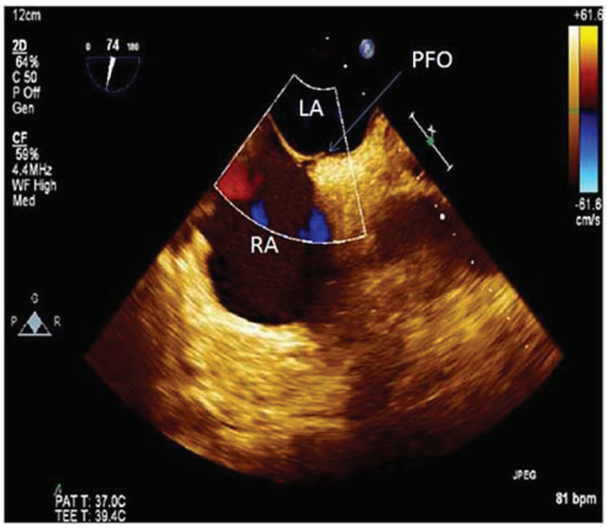

Figure 7 Transoesophageal echocardiogram showing small patent foramen ovale.

paradoxical embolisation was strongly suspected, especially in the context of very high pulmonary pressures. A transthoracic bubble echocardiogram (figure 6) was followed by a transoesophageal echocardiogram (TOE) with bubble study (figure 7), which confirmed the presence of a small PFO. During a bubble echocardiogram study, 10 ml of normal saline is agitated with a small volume of air to create bubbles and this is injected intravenously, usually followed by a valsalva manoeuvre, to visualise whether the bubbles cross from the right to left hand side of the heart, which would suggest a communication between the two.

A PFO closure was considered in our patient, but after multidisciplinary team discussion, it was decided that she would instead be managed with lifelong warfarin.

DISCUSSION

This case illustrates the phenomenon of myocardial infarction caused by paradoxical embolism through a PFO. Although rare, it should be considered in patients who present with acute coronary syndrome without significant underlying coronary artery disease.

Very few such cases, with concomitant pulmonary embolism, have been published in the literature.

Learning points

- Patients presenting with chest pain can have multiple co-existing pathological causes
- Apart from primary coronary artery disease, there are alternative mechanisms causing myocardial infarction
- These should be considered in patients with atypical presentations.

Competing interests None.

Patient consent Obtained.